

# PERK (EIF2AK3) Antibody (Center)

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP8150c

## Product Information

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Application	IHC-P, WB, E
Primary Accession	<a href="#">Q9NZJ5</a>
Other Accession	<a href="#">Q9Z1Z1</a> , <a href="#">Q9Z2B5</a>
Reactivity	Human
Predicted	Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB12501
Calculated MW	125216

## Additional Information

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Gene ID	9451
Other Names	Eukaryotic translation initiation factor 2-alpha kinase 3, PRKR-like endoplasmic reticulum kinase, Pancreatic eIF2-alpha kinase, HsPEK, EIF2AK3, PEK, PERK
Target/Specificity	This PERK antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide selected from the Center region of human EIF2AK3.
Dilution	IHC-P~~1:100~500 WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	PERK (EIF2AK3) Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

## Protein Information

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Name	EIF2AK3 {ECO:0000303 PubMed:10932183, ECO:0000312 HGNC:HGNC:3255}
Function	Metabolic-stress sensing protein kinase that phosphorylates the alpha

subunit of eukaryotic translation initiation factor 2 (EIF2S1/eIF-2-alpha) in response to various stress, such as unfolded protein response (UPR) (PubMed:[10026192](#), PubMed:[10677345](#), PubMed:[11907036](#), PubMed:[12086964](#), PubMed:[25925385](#), PubMed:[31023583](#)). Key effector of the integrated stress response (ISR) to unfolded proteins: EIF2AK3/PERK specifically recognizes and binds misfolded proteins, leading to its activation and EIF2S1/eIF-2-alpha phosphorylation (PubMed:[10677345](#), PubMed:[27917829](#), PubMed:[31023583](#)). EIF2S1/eIF-2-alpha phosphorylation in response to stress converts EIF2S1/eIF-2-alpha in a global protein synthesis inhibitor, leading to a global attenuation of cap-dependent translation, while concomitantly initiating the preferential translation of ISR-specific mRNAs, such as the transcriptional activators ATF4 and QRICH1, and hence allowing ATF4- and QRICH1-mediated reprogramming (PubMed:[10026192](#), PubMed:[10677345](#), PubMed:[31023583](#), PubMed:[33384352](#)). The EIF2AK3/PERK- mediated unfolded protein response increases mitochondrial oxidative phosphorylation by promoting ATF4-mediated expression of COX7A2L/SCAF1, thereby increasing formation of respiratory chain supercomplexes (PubMed:[31023583](#)). In contrast to most subcellular compartments, mitochondria are protected from the EIF2AK3/PERK-mediated unfolded protein response due to EIF2AK3/PERK inhibition by ATAD3A at mitochondria-endoplasmic reticulum contact sites (PubMed:[39116259](#)). In addition to EIF2S1/eIF-2-alpha, also phosphorylates NFE2L2/NRF2 in response to stress, promoting release of NFE2L2/NRF2 from the BCR(KEAP1) complex, leading to nuclear accumulation and activation of NFE2L2/NRF2 (By similarity). Serves as a critical effector of unfolded protein response (UPR)-induced G1 growth arrest due to the loss of cyclin-D1 (CCND1) (By similarity). Involved in control of mitochondrial morphology and function (By similarity).

#### Cellular Location

Endoplasmic reticulum membrane {ECO:0000250|UniProtKB:Q9Z2B5}; Single-pass type I membrane protein. Note=Localizes to the Localizes to endoplasmic reticulum membrane (By similarity). Also present at mitochondria-endoplasmic reticulum contact sites; where it interacts with ATAD3A (PubMed:39116259). {ECO:0000250|UniProtKB:Q9Z2B5, ECO:0000269|PubMed:39116259}

#### Tissue Location

Ubiquitous. A high level expression is seen in secretory tissues.

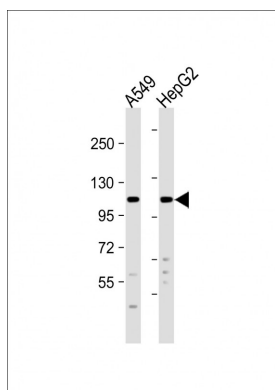
## Background

EIF2AK3, a member of the GCN2 subfamily of Ser/Thr protein kinases, phosphorylates the alpha subunit of eukaryotic translation-initiation factor 2 (EIF2), leading to its inactivation and thus to a rapid reduction of translational initiation and repression of global protein synthesis. This protein serves as a critical effector of unfolded protein response (UPR)-induced G1 growth arrest due to the loss of cyclin D1. It is proposed that perturbation in protein folding in the endoplasmic reticulum (ER) promotes reversible dissociation from HSPA5/BIP and oligomerization, resulting in transautophosphorylation and kinase activity induction. Expression of this Type I membrane protein is ubiquitous, with a high level expression in secretory tissues. Defects in EIF2AK3 are the cause of Wolcott-Rallison syndrome (WRS), also known as multiple epiphyseal dysplasia with early-onset diabetes mellitus. WRS is a rare autosomal recessive disorder, characterized by permanent neonatal or early infancy insulin-dependent diabetes and, at a later age, epiphyseal dysplasia, osteoporosis, growth retardation and other multisystem manifestations, such as hepatic and renal dysfunctions, mental retardation and cardiovascular abnormalities.

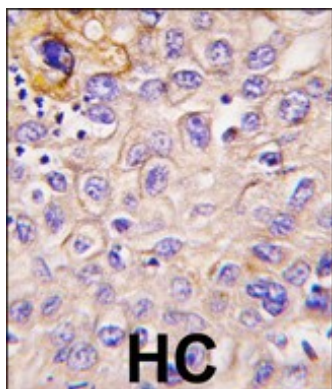
## References

- Delepine, M., et al., Nat. Genet. 25(4):406-409 (2000).  
 Shi, Y., et al., J. Biol. Chem. 274(9):5723-5730 (1999).  
 Sood, R., et al., Biochem. J. 346 Pt 2, 281-293 (2000).

## Images



All lanes : Anti-EIF2AK3 Antibody (Center) at 1:1000 dilution Lane 1: A549 whole cell lysate Lane 2: HepG2 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 125 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



Formalin-fixed and paraffin-embedded human hepatocarcinoma tissue reacted with EIF2AK3 antibody (Center)(Cat.#AP8150c), which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated.

## Citations

- [Regulation of SREBP-2 intracellular trafficking improves impaired autophagic flux and alleviates endoplasmic reticulum stress in NAFLD.](#)
- [HMGB1 induces endothelial progenitor cells apoptosis via RAGE-dependent PERK/eIF2α pathway.](#)

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